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 2/BI OR 216869-11-3/BI OR 216869-12-4/BI OR 216

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 DICTIONARY FILE UPDATES: 14 OCT 2001 HIGHEST RN 362009-74-3

TSCA INFORMATION NOW CURRENT THROUGH July 7, 2001

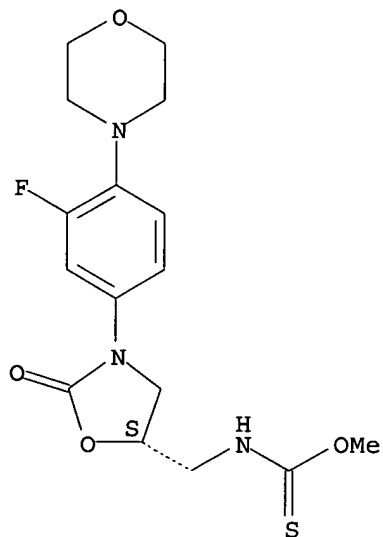
Please note that search-term pricing does apply when
 conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER see
 HELP CROSSOVER for details.

Calculated physical property data is now available. See HELP PROPERTIES
 for more information. See STNote 27, Searching Properties in the CAS
 Registry File, for complete details:
<http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf>

L10 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2001 ACS
RN 216868-99-4 REGISTRY
CN Carbamothioic acid, [[(5S)-3-[3-fluoro-4-(4-morpholinyl)phenyl]-2-oxo-5-oxazolidinyl)methyl]-, O-methyl ester (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C16 H20 F N3 O4 S
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



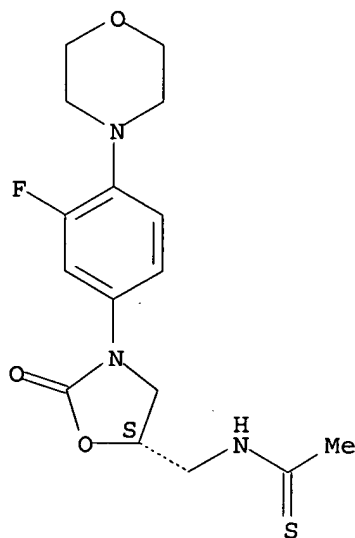
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5 REFERENCES IN FILE CA (1967 TO DATE)

5 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2001 ACS
RN 216868-57-4 REGISTRY
CN Ethanethioamide, N-[[(5S)-3-[3-fluoro-4-(4-morpholinyl)phenyl]-2-oxo-5-oxazolidinyl)methyl]- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C16 H20 F N3 O3 S
SR CA
LC STN Files: CA, CAPLUS, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

4 REFERENCES IN FILE CA (1967 TO DATE)
4 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L21 ANSWER 26 OF 46

MEDLINE

ACCESSION NUMBER: 96294739 MEDLINE

DOCUMENT NUMBER: 96294739 PubMed ID: 8698454

TITLE: Bacterially induced bone destruction: mechanisms and misconceptions.

AUTHOR: Nair S P; Meghji S; Wilson M; Reddi K; White P; Henderson B

CORPORATE SOURCE: Maxillofacial Surgery Research Unit, Eastman Dental Institute, University College London, United Kingdom.

SOURCE: INFECTION AND IMMUNITY, (1996 Jul) 64 (7) 2371-80. Ref: 137

Journal code: GO7; 0246127. ISSN: 0019-9567.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, ACADEMIC)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199609

ENTRY DATE: Entered STN: 19960912

Last Updated on STN: 19960912

Entered Medline: 19960904

AB Normal bone remodelling requires the coordinated regulation of the genesis

and activity of osteoblast and osteoclast lineages. Any interference with these integrated cellular systems can result in dysregulation of remodelling with the consequent loss of bone matrix. **Bacteria** are important causes of bone pathology in common conditions such as periodontitis, dental cysts, bacterial arthritis, and osteomyelitis. It is now established that many of the **bacteria** implicated in **bone diseases** contain or produce molecules with potent effects on bone cells. Some of these molecules,

such

as components of the gram-positive cell walls (lipoteichoic acids), are weak stimulators of bone resorption in vitro, while others (PMT, cpn60) are as active as the most active mammalian osteolytic factors such as cytokines like IL-1 and TNF. The complexity of the integration of bone cell lineage development means that there are still question marks over the mechanism of action of many well-known bone-modulatory molecules such as parathyroid hormone. The key questions which must be asked of the now-recognized **bacterial** bone-modulatory molecules are as follows: (i) what cell population do they bind to, (ii) what is the

nature

of the receptor and postreceptor events, and (iii) is their action direct or dependent on the induction of secondary extracellular bone-modulating factors such as cytokines, eicosanoids, etc. In the case of LPS, this ubiquitous gram-negative polymer probably binds to osteoblasts or other cells in bone through the CD14 receptor and stimulates them to release cytokines and eicosanoids which then induce the recruitment and

activation

of osteoclasts. This explains the inhibitor effects of nonsteroidal and anticytokine agents on LPS-induced bone resorption. However, other **bacterial** factors such as the potent toxin PMT may act by blocking the normal maturation pathway of the osteoblast lineage, thus inducing dysregulation in the tightly regulated process of resorption and replacement of bone matrix. At the present time, it is not possible to define a general mechanism by which **bacteria** promote loss of bone matrix. Many **bacteria** are capable of stimulating bone matrix loss, and the information available would suggest that each organism possesses different factors which interact with bone in

different

ways. With the rapid increase in antibiotic resistance, particularly with *Staphylococcus aureus* and *M. tuberculosis*, organisms responsible for much bone pathology in developed countries only two generations ago, we would urge that much greater attention should be focused on the problem of **bacterially** induced bone remodelling in order to define pathogenetic mechanisms which could be therapeutic targets for the development of new treatment modalities.

AB . . . Any interference with these integrated cellular systems can result in dysregulation of remodelling with the consequent loss of bone matrix. **Bacteria** are important causes of bone pathology in common conditions such as periodontitis, dental cysts, **bacterial** arthritis, and osteomyelitis. It is now established that many of the **bacteria** implicated in **bone diseases** contain or produce molecules with potent effects on bone cells. Some of these molecules, such as components of the gram-positive. . . action of many well-known bone-modulatory molecules such as parathyroid hormone. The key questions which must be asked of the now-recognized **bacterial** bone-modulatory molecules are as follows: (i) what cell population do they bind to, (ii) what is the nature of the. . . and activation of osteoclasts. This explains the inhibitor effects of nonsteroidal and anticytokine agents on LPS-induced bone resorption. However, other **bacterial** factors such as the potent toxin PMT may act by blocking the normal maturation pathway of the osteoblast lineage, thus. . . and replacement of bone matrix. At the present time, it is not possible to define a general mechanism by which **bacteria** promote loss of bone matrix. Many **bacteria** are capable of stimulating bone matrix loss, and the information available would suggest that each organism possesses different factors which. . . developed countries only two generations ago, we would urge that much greater attention should be focused on the problem of **bacterially** induced bone remodelling in order to define pathogenetic mechanisms which could be therapeutic targets for the development of new treatment. . .

L13 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1999:194131 CAPLUS

DOCUMENT NUMBER: 130:223265

TITLE: Preparation of

N-(2-oxothiazolidin-5-ylmethyl)thiourea
derivatives as antibacterial agents

INVENTOR(S): Yoshida, Toshihiko; Tokuyama, Ryukou; Tomita, Yayoi

PATENT ASSIGNEE(S): Hokuriku Seiyaku Co., Ltd., Japan

SOURCE: PCT Int. Appl., 137 pp.

CODEN: PIXXD2

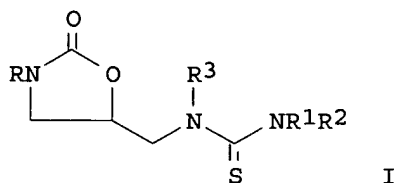
DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9912914	A1	19990318	WO 1998-JP4074	19980910
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
JP 11158164	A2	19990615	JP 1998-272500	19980909
AU 9890015	A1	19990329	AU 1998-90015	19980910
PRIORITY APPLN. INFO.:			JP 1997-265054	19970911
			WO 1998-JP4074	19980910
OTHER SOURCE(S):		MARPAT 130:223265		
GI				



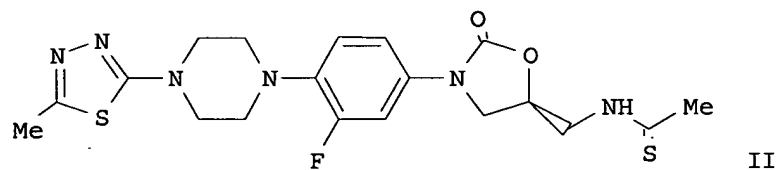
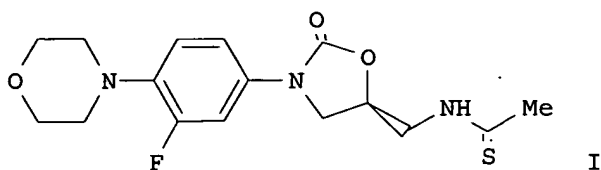
AB Antimicrobial thiourea derivs. of general formula (I) or salts thereof: (wherein R1, R2, and R3 are each hydrogen, alkyl, cycloalkyl, nitrogen-protecting group, alkoxy-carbonylalkyl or the like; and R is Ph which may be substituted by halogeno, hydroxyl, mercapto, amino, cyano, nitro, carboxyl, carbamoyl, alkyl, cycloalkyl, alkoxy, alkylamino, alkanoyl, arylcarbonyl, aryl, aralkyl, aryloxy, cycloalkyloxy contg. a hetero-atom as a ring atom, a satd. heterocyclic group or the like) are prepd. Also claim is an antibacterial agent, in particular against gram pos. bacteria, contg. I as the active ingredient. These thiourea derivs. exhibit excellent antibacterial activity against not only normal bacteria but also resistant strains of bacteria, e.g. methicillin-resistant Staphylococcus aureus (MRSA). Thus, addn. reaction of (R)-[2-oxo-3-[4-(thiomorpholin-4-yl)phenyl]oxazolidin-5-yl]methyl isothiocyanate with NH3 in MeOH at room temp. for 9 h gave I [R = 4-(thiomorpholin-4-yl)phenyl, R1 = R2 = R3 = H]. I [R =

3-fluoro-4-(pyrrolidino-1-yl)phenyl, R1 = R2 = R3 = H] showed min. inhibitory concn. of 0.39 .mu.g/mL against MRSA HPC1336 and Enterococcus faecalis HPC948 and HPC975.

REFERENCE COUNT: 8
 REFERENCE(S): (1) Bayer Ag; JP 09316073 A CAPLUS
 (3) Bayer Ag; DE 19649095 A1 CAPLUS
 (4) Bayer Ag; US 5792765 A CAPLUS
 (5) Bayer Ag; EP 789025 A1 1997 CAPLUS
 (6) Bayer Ag; EP 789026 A1 1997 CAPLUS
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2001 ACS
 ACCESSION NUMBER: 1998:794995 CAPLUS
 DOCUMENT NUMBER: 130:38373
 TITLE: Preparation of thiocarbonyloxazolidinones as antibacterial agents
 INVENTOR(S): Hester, Jackson B. Jr; Nidy, Eldon George; Perricone, Salvatore Charles; Poel, Toni-jo
 PATENT ASSIGNEE(S): Pharmacia & Upjohn Company, USA; Hester, Jackson B., Jr.
 SOURCE: PCT Int. Appl., 118 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 2
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9854161	A1	19981203	WO 1998-US9889	19980518
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9874883	A1	19981230	AU 1998-74883	19980513
EP 984947	A1	20000315	EP 1998-922303	19980518
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9815518	A	20001121	BR 1998-15518	19980518
NO 9905846	A	20000128	NO 1999-5846	19991129
FI 9902555	A	19991130	FI 1999-2555	19991130
PRIORITY APPLN. INFO.:			US 1997-48342	P 19970530
			WO 1998-US9889	W 19980518
OTHER SOURCE(S):			MARPAT 130:38373	
GI				



AB Chiral title compds. AGCH2NHCSR [A is (un)substituted Ph, indoliny1; G is 2-oxo-5-oxazolidiny1; R is H, NH₂, alkyl, cycloalkyl, etc.] or pharmaceutical acceptable salts are prepd., from amines with Lawesson's Reagent or 1,1'-thiocarbonyldi-2(1H)-pyridone, as antibacterial agents. Title compds. I and II were tested in vitro by std. agar diln. method.

REFERENCE COUNT:

8

REFERENCE (S) :

- (1) Bayer AG; EP 0789025 A 1997 CAPLUS
- (2) Bayer AG; DE 19601264 A 1997 CAPLUS
- (3) E I du Pont de Nemours and Company; EP 0127902 A 1984 CAPLUS
- (4) E I du Pont de Nemours and Company; EP 0184170 A 1986 CAPLUS
- (5) Pharmacia & Upjohn Company; WO 9807708 A 1998 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT